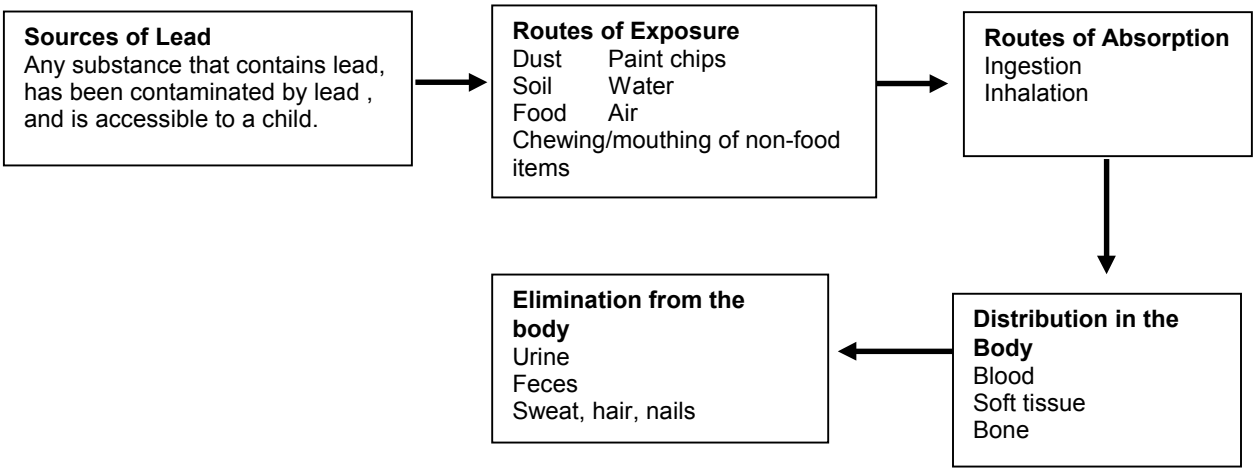
Lead in the Human Body

Just as the sources of lead exposure in children vary from adults, so does the way a child's body metabolizes and is affected by lead. Figure 4.1 shows the path lead takes in a child's body from exposure (usually through hand-to-mouth activity) to elimination.

Figure 4.1
Lead Sources and Routes of Exposure



Absorption of Lead in Children's Bodies

The primary site of absorption of lead in children is the gastrointestinal tract. Ingestion through hand-to-mouth activity is the primary manner in which children introduce lead into their bodies. Children absorb up to 50% of the lead they ingest, about five times as much as adults. Gastrointestinal absorption is enhanced by a fasting state, iron or calcium deficiency, and/or high fat diets. No feedback mechanism causes a decrease in the absorption of ingested lead once levels become elevated.

Lead is absorbed rapidly through the lungs when inhaled. Up to 70% of inhaled lead is absorbed, depending on particle size. The primary source of inhaled lead had been emissions from automobiles using leaded gasoline. Since the decrease in leaded gasoline levels, the total amount of lead inhaled by children is small compared to the amount ingested.

Absorption of lead through the skin is minimal. Lead poisoning in children through dermal exposure is rare, primarily because children's contact with these elements is limited.

Distribution of Lead in a Child's Body

Once absorbed into the child's system, lead is distributed in three body systems: blood, soft tissue, and bone. The concentration and mobility of lead within each compartment varies (see Figure 4.2)

The blood lead level (BLL) is the most direct measurement of body lead, although it represents only about 5-10% of the total body lead burden. Once in the blood, up to 99% of lead is bound to erythrocytes and cannot diffuse across cell membranes.

Approximately 1-10% is bound to microligands in the plasma. It is this pool that is capable of crossing cell membranes and therefore can become biologically active. Because lead is found primarily in the red blood cell rather than plasma, there are implications when collecting capillary blood lead samples. If the finger is squeezed too hard, a blood sample may be obtained that is higher in plasma, resulting in a BLL that is falsely low. Lead readily binds to fetal hemoglobin.

Figure 4.2 Distribution of Lead in the Body

	Blood	Soft Tissue	Bone
Half-life	35 days	40 days	Spongy (pelvis, ribs, skull): 3-5 years Cortical (midtibia, midfemur): 30 years
% total body burden	5-10%	10-20%	70-85%

Source: Lead Poisoning in Childhood, S. Pueschel, J. Linakis, A. Anderson

About 10-20% of lead retained in the body is stored in soft tissues such as kidney, liver, bone marrow, and brain. It is in these sites where lead has the most toxic effects to children. The toxic effects vary inversely with the age of the poisoned child: the younger the child, the more vulnerable the sites are to lead exposure. The extent of damage to soft tissues is related directly to the amount and duration of exposure: the longer the exposure, the more severe the effects. In other words, the younger the child and the longer the exposure, the greater the severity of the effects is likely to be.

The remainder of lead retained by the body is stored in the bones. The half-life of lead in bone can be up to 30 years, and throughout that time it seeks to create a steady-state with blood lead. As the BLL drops due to chelation and/or decreased exposure, lead migrates from the bone to blood, and may be the cause of a prolonged elevated blood lead level. Bone-to-blood migration may also occur during pregnancy and lactation in women with high bone lead levels, which may have developed in early childhood.

Elimination of Lead from Children's Bodies

An estimated 60% of absorbed lead is eliminated from the body. The primary route of elimination is through the kidney, followed by feces, hair and nail growth, and sweat loss

In animal studies comparing lead metabolism in infant and adult rats, lead was cleared from the blood much more slowly, and localized in the brain to a greater degree in infant rats.

Primary Effects of Lead Toxicity in Young Children

The Centers for Disease Control & Prevention identifies lead as the number one environmental health threat to young children. Lead toxicity can have an adverse affect on virtually every system in the body. The result of lead toxicity can be seen in the peripheral nervous, hematopoietic, renal, and gastrointestinal systems. It effects the regulation of vitamin D, and the growth, hearing, and cognitive development of a young child. Most importantly, it can cause irreversible damage to the central nervous system. At very high levels, lead exposure can cause seizures, coma, and death.

Continued research on lead toxicity in children has caused a rapid decrease in the blood lead levels of concern for children. As recently as the early 1960's, the level of concern for children was 60μg/dL. In 1985 that level was lowered to 25μg/dL, and to the current

level of 10μg/dL in 1991. That decrease was prompted by an acceptance of widespread research that showed damage from lead at BLLs as low as 10μg/dL to the central nervous system of young children, causing developmental delays, lower IQ, hyperactivity, learning disabilities, behavioral problems, and school failure. Physical effects noted at this level include impaired hearing, slowed growth, and nephropathy. In the fall of 2002, two national panels began to examine the research on the negative affects of even small amounts of lead on children's intelligence. They will make recommendations on whether the CDC should lower the "acceptable level of lead in a child's blood (from 10μg/dL set in 1991).

Animal research on the effects of lead on brain structure and function demonstrate multiple cellular and synaptic disruptions caused by lead. These disruptions in cellular anatomy and neurotransmitter systems can be noted by the breakdown in their function of modulating emotional response, memory, learning and visual-spatial relationships. Studies have persistently shown a correlation between low-level lead exposure during early brain development and deficits in neurobehavioral-cognitive performance that manifest later in childhood. The effects demonstrated in several longitudinal studies have been consistent across cultures, racial/ethnic groups, and social/economic class.

In the first research that specifically looked at a large number of children with BLLs known to be <10μg/dL, deficits in cognition and academic abilities associated with lead exposure have been noted. Adjusting for factors known to have an impact on these outcomes, children with BLLs ≥3μg/dL demonstrated impairment in cognitive, memory and visual-spatial skills. The adverse effects of lead on reading and other language-based abilities was significant, as these are potent predictors of academic achievement and anti-social behavior. This study further supports the research that has indicated no threshold for lead toxicity.

Research indicates that lead exposure during the first 3 years of life has the most damaging and long-lasting effects. At this stage, the child's developing brain is most vulnerable to toxic exposures. One study indicated that a stronger predictor of school-age intellectual function was elevated blood lead at any level at an early age, than even higher BLLs in older children.

A publication by the Wisconsin Council on Children and Families on brain development and the impact of environmental factors can be found at the end of this chapter. Entitled "Great Beginnings: The First Years Last Forever", the paper summarizes early brain growth, critical periods of development, and how environmental factors during these stages can affect long-term growth and development of the child. For more information on brain development, see the Council's website at www.wccf.org.

Prenatal lead exposure has been associated with increased risk of pre-term delivery, reduced birth weight, and reduced performance on neurological testing. For children whose subsequent lead exposure is low and who receive developmentally appropriate stimulation, there is evidence that the damaging neurological effects associated with prenatal exposure may be reversible.

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